SPECIFICATION AMENDMENTS

Replace paragraph [0004] on page 1 with the following:

[0004] *S*-[2-([[1-(2-ethylbutyl)cyclohexyl]carbonyl]amino)phenyl] 2-methylpropanethioate has been shown to be an inhibitor of CETP activity in humans (de Grooth et al., *Circulation, 105,* 2159-2165 (2002)) and rabbits (Shinkai et al., *J. Med. Chem., 43,* 3566-3572 (2000); Kobayahi et al., *Atherosclerosis, 162,* 131-135 (2002); and Okamoto et al., *Nature, 406(13),* 203-207 (2000)). *S*-[2-([[1-(2-ethylbutyl)cyclohexyl]carbonyl]amino)phenyl] 2-methylpropanethioate has been shown to increase plasma HDL cholesterol in humans (de Grooth et al., *supra*) and in rabbits (Shinkai et al., *supra*; ~~Kobayahi~~ Kobayashi et al., *supra*; Okamoto et al., *supra*). Moreover, *S*-[2-([[1-(2-ethylbutyl)cyclohexyl]carbonyl]amino)phenyl] 2-methylpropanethioate has been shown to decrease LDL cholesterol in humans (de Grooth et al., *supra*) and rabbits (Okamoto et al., *supra*). Additionally, *S*-[2-([[1-(2-ethylbutyl)cyclohexyl]carbonyl]amino)phenyl] 2-methylpropanethioate inhibits the progression of atherosclerosis in rabbits (Okamoto et al., *supra*). *S*-[2-([[1-(2-ethylbutyl)cyclohexyl]carbonyl]amino)phenyl] 2-methylpropanethioate, as well as methods of making and using the compound, are described in U.S. Patent 6,426,365.

Replace paragraph [0021] on page 5 with the following:

[0021] While not wishing to be bound by any particular theory, it is hypothesized that within the body of a patient, Compound I is hydrolyzed in plasma, the liver, and/or the small intestine to form *S*-[2([[1-(2-ethylbutyl)cyclohexyl]carbonyl]amino)phenyl] thiol (herein referred to as Compound II). It is known that low molecular weight thiol components (i.e., R-SH), such as cysteine and glutathione, and high molecular weight thiol components (i.e., Prot-SH), such as peptides and proteins (e.g., enzymes and cell membranes), exist in the body as mixed disulfides containing an oxidized disulfide bond (S-S bond) between or within the molecule (see, e.g., ~~Shimade~~ Shimada et al., *J. Chromatogr. B, 659,* 227 (1994)). Therefore, it is hypothesized that within the body of a patient, Compound II is conjugated with low or high molecular weight thiols to yield mixed disulfides or to yield dimers of Compound II. Since these forms are in an oxidation-reduction equilibrium with each other via Compound II, all of these forms, as well as Compound II, are collectively, but not exclusively, considered and referred to hereafter as the active form of Compound I. The following scheme depicts the above-described hypothesis.